SILICOSIS AND ASBESTOSIS

THE LANCET

Silicosis and Asbestosis

BY VARIOUS AUTHORS

EDITED BY

A. J. LANZA, M.D.

ASSISTANT MEDICAL DIRECTOR, METROPOLITAN LIFE INSURANCE COMPANY;
CHAIRMAN, INDUSTRIAL HYGIENE COMMITTEE OF THE NEW YORK
TUBERCULOSIS AND HEALTH ASSOCIATION

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PREFACE

THE industrial age has complicated the scheme of life in many ways, some of them directly affecting the public health ; among these are the diseases due to the inhalation of certain kinds of atmospheric dusts arising directly from industrial operations. Ill effects due to dust contamination of the atmosphere have been recognized from earliest times.

This book has for its purpose the presentation of the medical and public health aspects of silicosis and asbestosis, two definitely industrial dust diseases. Silicosis looms larger in the picture than asbestosis because the former is much more widely spread, affects a much greater number of the industrial population, has been recognized and studied longer and, consequently, more is known about it.

The form of the generic term pneumoconiosis, rather than pneumonoconiosis or pneumonokoniosis, has been adhered to throughout the book. The International Labour Office and both British and American editors have adopted it as preferable.

The editor wishes to express special thanks to Miss C. M. Bresnan for reading proof and for making the index and to acknowledge with gratitude the help received from many sources and from many individuals. To them and particularly those working in the field of industrial hygiene, this book is dedicated.

A. J. LANZA, M.D.

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CONTRIBUTORS

A. J. LANZA, M.D.

Assistant Medical Director, Metropolitan Life Insurance Company ; Chairman, Industrial Hygiene Committee of the New York Tuberculosis and Health Association.

Formerly *in Charge of the Office of Industrial Hygiene, United States Public Health Service ; Chief Surgeon of the United States Bureau of Mines ; Adviser on Industrial Hygiene to Commonwealth Government of Australia ; Special Staff Member of the International Health Board of the Rockefeller Foundation ; Medical Consultant of General Motors Corporation.*

R. R. SAYERS, A.M.,M.D.

Senior Surgeon, United States Public Health Service ; Chief, Division of Industrial Hygiene, National Institute of Health.

Formerly *Chief Surgeon, United States Bureau of Mines.*

EUGENE P. PENDERGRASS, M.D.

Professor of Radiology, Medical School, University of Pennsylvania ; Professor of Radiology, Graduate School of Medicine, University of Pennsylvania ; Associate Director, Department of Radiology, Hospital of the University of Pennsylvania.

S. ROODHOUSE GLOYNE, M.D.,D.P.H.

Pathologist, Pathological Laboratories and Research Institute, The London Chest Hospital.

LEROY U. GARDNER, M.D.

Director, Saranac Laboratory for the Study of Tuberculosis.

E. L. MIDDLETON, M.D.,D.P.H.

H. M. Medical Inspector of Factories, London, England.

INTRODUCTION

PATHOLOGICAL conditions of the lungs, due to the inhalation of excessive quantities of dust in certain occupations, have been known to exist for many centuries. It is also true that the frequency of infection, particularly pulmonary tuberculosis, as a complication has been realized for a long time. Pneumoconiosis is a comprehensive term, designating the effects upon the lungs of the inhalation of excessive quantities of dust, manifested by structural changes in the lung tissue and entirely distinct from the action of poisonous dust, such as lead or mercury, in which case the lungs act merely as the point of entrance into the body without definite local influence. At first it was associated chiefly with mining but with the advancement of the industrial age, dusty industries multiplied.

It is customary to speak of the dust inhalation effect upon the lung tissue by the name of the chief constituent of the dust breathed, as silicosis when silicon dioxide (SiO_2) is the principal ingredient and asbestosis when asbestos is the offending agent. Silica dust, when the particles are sufficiently small, has the peculiar property of inducing a fibrosis of the lung tissue. This effect is characteristic, as is the resulting roentgenologic appearance. In asbestosis, the pathological process is quite different as is the roentgenologic appearance. These matters are thoroughly dealt with in the text. There is still, however, much to be learned with respect to both conditions. We know the results of the inhalation of these dusts but we are not sure how they are produced.

There are two ways in which the pulmonary infections, tuberculosis and pneumonia, may affect or complicate pneumoconiosis. First, there may be infection before the exposure

to dust. It is an undecided question whether such infection can so change the pulmonary structure that the later dust effect is more pronounced than it would otherwise be. There seems to be reason to believe that such an enhancement is to be expected, especially if the previous infection were tuberculosis, but this must be left to the histopathologist for final proof. Secondly, do such infections upon an already dusted lung become more severe or aggravate the dust effect or otherwise change its character? There is even more evidence that any of these effects may result but here again the histopathologist must be called upon to tell us the exact truth. The first intimation that occurred to us that the character of a silicotic lesion could be so changed was when we were searching for an explanation as to why certain individuals with silicosis showed massive consolidations of fibrosis in the lungs and others in the same occupation and apparently exposed to the same amount of dust of the same variety showed a more evenly diffused nodular fibrotic process. It was a great satisfaction to hear a well known pathologist say that a comparatively recent infection like lobar pneumonia might be the answer but that at present there was no definite proof of such theory being correct.

It was gratifying to learn that most of us were thinking along similar lines. That most cases of silicosis die of an intercurrent infection, especially tuberculosis and possibly pneumonia, is evidence that silicosis may exert some effect upon the progress of some pulmonary infections. It has not been proven as yet that the same is true of asbestosis.

A comprehensive knowledge of the effects upon the lungs of excessive inhalations of dust is not to be quickly gained. The combined efforts of the histologist, pathologist, clinician, roentgenologist, chemist, physicist and other technological experts are necessary. The legal profession deserves a certain share of commendation for stimulating the study of

occupational dust diseases and has made a thorough knowledge of pneumoconiosis a necessity among many of the medical men. Were it not for the demand for exactness from outside the medical profession, it is difficult to say how soon curiosity or medical research would have achieved our present accuracy of knowledge with respect to the etiology, symptoms, and pathology of pulmonary dust diseases and their modification by certain infections.

A large number of us have, no doubt, taken up the study of pneumoconiosis from the standpoint of interest. Following the experience with silicosis in South Africa, starting several decades ago, text books for the medical student simply mentioned it in a more or less cursory manner as an uncommon condition. We are sure of the necessity of many angles of approach to the subject. The first comprehensive report of silicosis in the United States was made about twenty years ago by one of the authors. It described what was occurring among miners in the south west. Almost simultaneously and without any knowledge of this report, it occurred to a small group of medical men in the east that the best way to study the differences in the appearances of trunk shadows in the roentgenograms of the lungs might be to study the chests of persons engaged in various dusty occupations. The main point learned in this investigation was that organic dusts do not induce lung changes but it further stimulated the study of the effects of inorganic dusts, particularly silica, and later, asbestos. This study has continued up to the present time.

Some of the authors of this book were among the pioneers in silicosis and asbestosis ; they have blazed the trail for future investigations. It can be truthfully stated that those who studied these occupational diseases in North America and the British Empire have always had the good of the affected workers in mind. Their researches have saved life and have made many occupations safer for the worker.

HENRY K. PANCOAST, M.D.

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